Vitamin E COVID-19 Platelet Information

Vitamin E Covid-19 Platelet Information
by Anthony of Boston

The number one cause of death for those suffering with severe COVID-19 is respiratory failure from Acute Respiratory Distress Syndrome(ARDS). Research has found that ARDS is closely linked to coagulation activation. This increased thrombotic risk among COVID-19 patients has yet to be fully explained. This is due to the increased prevalence of thrombocytopenia or low platelet counts among those suffering with severe COVID 19. While a low platelet count would indicate a risk for bleeding, most COVID-19 deaths are linked to higher thromboembolism risk. Some studies have linked a higher mean platelet volume(MPV) to COVID-19 severity. This has confused researchers for some time. Studies have concluded that both increased mean platelet volume (MPV) and decreased platelet count should serve as biomarkers for COVID-19 disease severity. Platelet count is the number of platelets circulating our blood, while Mean Platelet Volume (MPV) indicates the size of the platelets. MPV is also linked to the activity of the platelets. Higher MPV is associated with higher reactivity of the platelets. Larger platelets are considered more reactive. While blood thinners like aspirin and warfarin can reduce platelet count, they do little to affect the size of the platelets. (Aspirin is still more effective at lowering MPV then Warfarin). In fact Warfarin, which has been used in treatment protocols for COVID-19 patients, has been found in a study to both lower platelet count and increase MPV.
https://bmccardiovascdisord.biomedcentral.com/articles/10.1186/s12872-015-0047-8

Researchers also found that the odds of the mean platelet volume being high in severe COVID-19 was almost 60%. With clot risk being higher than bleeding risk among severe COVID-19 cases, we can presume that high mean platelet volume(MPV) should be singled out from low platelet count as the bio-marker for severe COVID-19 mortality risk from

respiratory failure. Low platelet count on the other hand should serve as a bio-marker for severe COVID-19 mortality risk from gastrointestinal(GI) bleeding. This leaves doctors having to navigate a fine line between the two in severe cases. This would help us infer that treatment geared toward reducing the size and hyperactivity of the platelets should serve as a means of alleviating the respiratory distress, but at the same time raise GI bleeding risk. During circulation, platelets are reactive to various stimuli. A high MPV with a low platelet count indicates that the platelets, even though low in number, are going into circulation very quickly and raising the risk of blood clots. Vitamin E has been shown to reduce both platelet count and platelet reactivity.

Gastrointestinal bleeding occurs in about 2-3% of ARDS COVID-19 cases. It is independently associated with a greater mortality risk and prolonged hospital stay. However, a few case studies have shown that the onset of Gastrointestinal bleeding in ARDS patients was preceded by an improvement in respiratory symptoms. I hypothesize that a greater risk of gastrointestinal bleeding is associated with a lower risk of respiratory distress. Even though the ARDS patient would have suffered from gastrointestinal bleeding issues, one can still observe the fact that severe respiratory symptoms did improve just before the onset of the gastrointestinal bleeding. In severe COVID-19, there is a fine line to walk between eliminating the risk associated with thrombosis and raising the risk associated with GI bleeding through application of blood thinning medications.

Here is a case of someone whose respiratory symptoms improved just before Gastrointestinal bleeding. https://www.peertechzpublications.com/articles/AGGR-5-124.php

Here is another case study of GI bleed in someone diagosed with COVID-19:
“Our case shed light on an unusual presentation of COVID-19. The patient had a GI bleed, which was evidently caused by

warfarin toxicity. However, elevated INR seemed to have played a protective role in this patient from the respiratory manifestations of COVID-19."
https://www.cureus.com/articles/52310-an-unusual-case-of-gastrointestinal-bleeding-in-a-patient-with-covid-19

This is a case study in Wuhan of someone who was critically ill with COVID-19, but died from Gastrointesitinal bleeding.
"We presented a critically ill patient with COVID-19 who progressed rapidly with ARDS, and ultimately died due to massive GIB even after improvement of respiratory status."
https://bmcgastroenterol.biomedcentral.com/articles/10.1186/s12876-020-01458-x

It would be very interesting if more data can confirm an improvement of respiratory symptoms before onset of Gastrointestinal(GI) bleeding issues. In the most severe cases, doctors may be able to improve prognosis of a severely ill ARDS COVID-19 patient by bringing INR levels beyond the therapeutic range. This would increase a severely ill ARDS COVID-19 patient's risk factors for GI bleeding, but at the same time raise the odds of alleviating their respiratory distress....if my hypothesis is correct. This fine line where GI bleeding issues would arise would have to be met with Vitamin K or some sort of pro-coagulant intervention in a timely manner in order to circumvent death. INR measures the time it takes for the blood to clot. A higher INR means that the blood takes a longer time to clot. Blood thinners tend to raise raise INR levels. It was found in a number of studies that a higher INR is associated with disease severity and non-survival in COVID-19. However, it is quite possible that ARDS disease progression out pacing INR could be the reason for higher mortality outcomes. By raising the INR beyond the therapeutic range in severe COVID-19 cases, one may be able to reduce the platelet size or mean platelet volume(MPV) and thus improve respiratory symptoms. This may be the reason why blood thinners like Aspirin and Warfarin haven't been associated with decreasing MPV—the dosages may not have been high enough. Even

though they inhibit platelet aggregation, they haven't been found to fully inhibit platelet activation at the dosage level used in tests.
See https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5906253/

The therapeutic INR range is 2.0-3.0. When myocardial infraction still occurs, the therapeutic range is increased to 2.5 – 3.5 as a secondary prevention protocol with Warfarin. Studies have shown that going beyond 4.0 shows no therapeutic benefit but increases the risk for bleeding. However, for the most severe cases of ARDS COVID-19, the range may have to be raised to 4.0 or higher in order to lower the mean platelet volume and improve respiratory symptoms.

It's possible that raising the dosages and GI risk could effect platelet activation and platelet size. Gastrointestinal bleeding has been associated with a lower mean platelet volume. Therefore increased risk of GI bleeding should also be associated with a decreasing MPV and a decreased reactivity of the platelets.

Vitamin E's anti-viral and anti-coagulant properties could be used to raise INR levels in severe COVID -19 cases. But should not be applied with current anti-coagulation medication like warfarin, as this could provoke uncontrollable coagulopathy. Aspirin, however, may be an exception. There are studies that indicate that Vitamin E when combined Aspirin improves the efficacy of Aspirin. https://pubmed.ncbi.nlm.nih.gov/11940487/
Aspirin is also more effective than warfarin at reducing MPV. I presume that ARDS disease progression would require a higher dosage of vitamin E--enough to raise the risk factors for gastrointestinal bleeding so that respiratory distress can be alleviated. Slightly raising the INR therapeutic range to 4.0 may suffice as a safer early measure. The patient would later have to be treated with Vitamin E antagonists in order to combat the risk of Gastrointestinal bleeding. Vitamin K is usually the standard for pro-coagulation therapies and is also considered an antagonist against against the anti-coagulation activities of

Vitamin E COVID-19 Platelet Information

Vitamin E.

Vitamin E has been shown to inhibit both platelet aggregation and platelet activation.
https://www.aafp.org/afp/1999/0901/p895.html

Vitamin E COVID-19 Platelet Information

Bibliography

https://bmccardiovascdisord.biomedcentral.com/articles/10.1186/s12872-015-0047-8

Mean Platelet Volume May Represent a Predictive Parameter for Overall Vascular Mortality and Ischemic Heart Disease Georg Slavka, Thomas Perkmann, Helmuth Haslacher, Stefan Greisenegger, Claudia Marsik, Oswald F. Wagner, Georg Endler

https://www.thelancet.com/journals/eclinm/article/PIIS2589-5370(20)30383-7/fulltext

https://pubmed.ncbi.nlm.nih.gov/12152667/

The effects of vitamin E on platelet activity in b-thalassaemia patients Supeenun Unchern,1 Narumol Laoharuangpanya,2 Noppawan Phumala,1 Pornpan Sipankapracha,3 Pensri Pootrakul,3 Suthat Fucharoen,3 Wanchai Wanachivanawin4 and Udom Chantharaksri1 1Department of Pharmacology, Faculty of Science, Mahidol University, 2 Laboratory of Pathobiology, Chulaborn Research Institute, 3 Thalassemia Research Centre, Institute of Science and Technology for Research and Development, Mahidol University, and 4 Department of Medicine, Faculty of Medicine, Mahidol University, Bangkok, Thailand Received 5 June 2003; accepted for publication 20 August 2003

https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.3001109

https://pubmed.ncbi.nlm.nih.gov/21247392/

https://www.news-medical.net/news/20210722/Can-platelet-size-predict-severity-and-mortality-in-COVID-19.aspx

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5906253/